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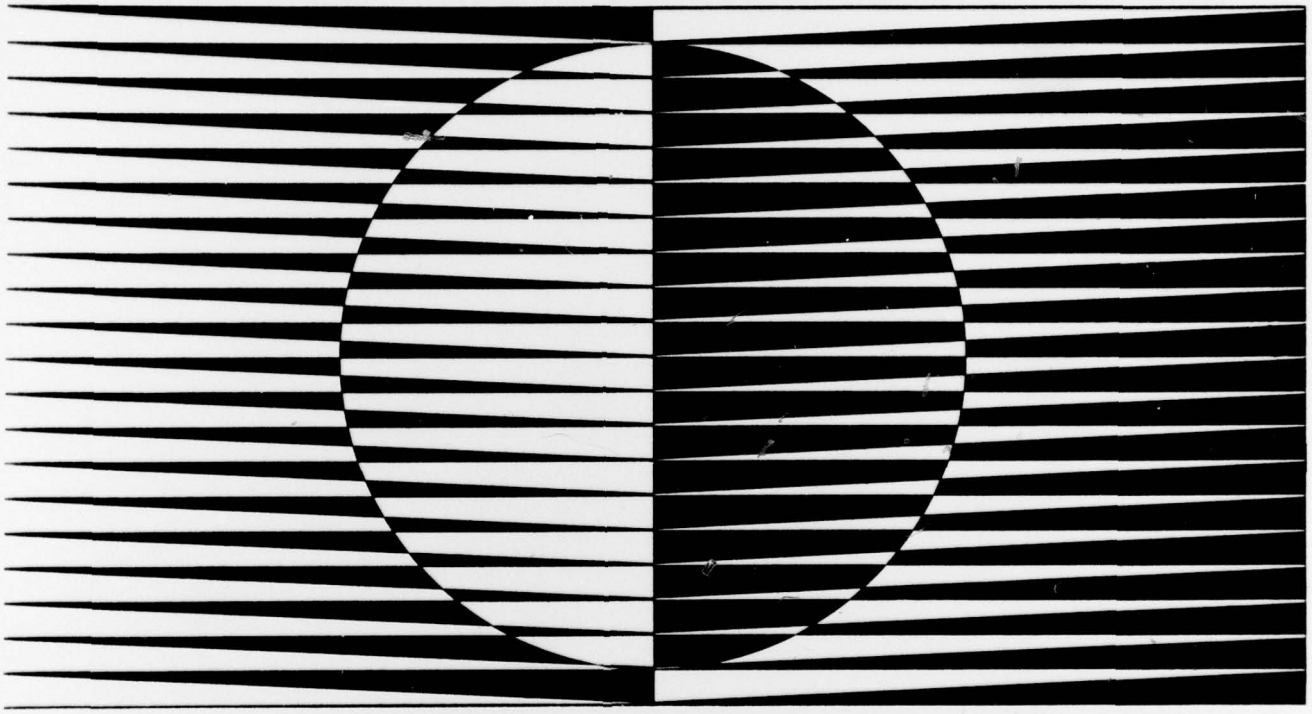

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SLEEP DEPRIVATION IN HUMAN SUBJECTS: A REAPPRAISAL

by

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Abstract

Some recent studies on sleep loss reveal significant improvements in biochemical, physiological and psychological methods of assessment. Critical examination of these studies indicates altered research emphases, some newly developed research concepts, and a new awareness of the methodological complexity involved in the seemingly simple research tool, sleep deprivation. The present paper suggests that sleep deprivation research will remain important in advancing our understanding of the impact of sleep loss, and of the mechanisms for the need of sleep.

Introduction

THE EFFECTS OF SLEEP DEPRIVATION (SD) IN HUMANS have been studied extensively. Many reviews have appeared, and some are given in the references for this paper (14, 15, 17, 24, 26, 29, 31, 34, 36, 42, 53, 59, 60, 63). In addition, two symposia directly concerned with SD have been held recently (12, 65).

This paper has been prepared to provide a multidisciplinary view of SD effects. Many past reviews have been unsatisfactory and even misleading, as their coverage has been so limited that they have failed to evaluate the effects of SD as an integral whole. The only exceptions to these narrowly-based highly-specialized reviews are reports by Jovanović et al. (34), Levi (36) and Wilkinson (60): they have examined the effects of SD across many disciplines, *e. g.* biochemistry, physiology and psychology, covering the literature up to 1971. Investigators who rely heavily and uncritically on specialized reviews face the risk of underestimating the impact of SD on humans. For example, short-term loss of sleep, *i. e.* sleep loss of less than 40 hours, has been shown definitely *not* to affect the human cognitive and memory functions which are necessary to perform assigned tasks under field conditions (31). However, this does not imply either that the effects of SD are completely innocuous or that there is little point in continuing the field studies to determine the undesirable consequences of SD. I can easily imagine a field condition where men would be exposed to several short-term SDs, one after the other, without adequate opportunity for full recovery from the effects of each preceding episode of SD, resulting in possible stress-related physical ailments. Indirect evidence for assuming the occurence of such dire operational consequences due to short-term SD already exists in the current literature (9, 10, 21, 22, 26, 36, 44, 48, 49). Thus, when the study of the effects of SD is not limited to the effects on task performance alone, but is extended to include the much broader interdisciplinary framework of the individual's health, can we arrive at the adequate conclusion that even a short-term SD could be hazardous. Emphasis on the multidisciplinary approach does not imply that the specialized reviews are useless and inaccurate. They do give us a clear, but perhaps too simplified view of the effects of SD on humans within a set boundary. As the review by Johnson and Naitoh (31) has indicated, it is indeed true that short-term SD does not seriously interfere with the completion of assigned tasks under field conditions. Dramatic support of their view comes from participants in single-handed transatlantic yacht racing (6). These participants functioned well enough to complete the 3,000 mile ocean crossing, despite having to wake themselves up at hourly or even more frequent intervals throughout 20—58 day yacht race. I do not dispute the observed robustness of certain aspects of the task performances under the field conditions. However, I wonder if a multidisciplinary approach would have revealed biochemical, physiological and long-term health "cost" from undertaking such an arduous adventure.

In the present paper, *five research trends* in recent studies of SD will be examined to show the extent of progress and also to highlight unresolved critical problems. The *first research trend* is the effort to evaluate studies on SD in the context of sleep research. Here sleep loss research is closely woven into the fabric of general sleep research. The *second research trend* is the renewed interest in obtaining non-performance measures of the effects of SD. This trend represents the most clear-cut departure from most of the past SD researches in which the anchoring point was invariably related to task performance. The *third trend* is the increase in field studies. The *fourth trend* places emphasis on individual differences. Finally the *fifth trend* is reflected in the increase of medically oriented research.

I. Sleep Deprivation and Sleep Research

Research on SD is as old as that which explores the nature of sleep, and there used to be a very close link between these two fields of scientific inquiry. However, nowadays, we are losing the close interaction between sleep researches and SD studies. The researchers of SD have developed their own specialized field almost independent of sleep research, intensively cataloging the impact of SD on task performances, physiological variables and others. For some SD researchers, SD has become merely a tool used to change the experimental subject's body and mind. Reporting these changes is a major obsession of some SD studies, leading them to, euphemistically speaking, an empiricism which in fact is to confirm repeatedly a truism: *SD makes humans and animals sleepy.*

Recently Dement (14), Dement and Mitler (15) and Rechtschaffen (53) have raised methodological questions concerned with SD as seen in the context of sleep research.

With respect to total SD, Dement (14) contends that this can not be done with human subjects. They invariably slip in and out of "microsleep" during the period of supposedly "total" SD. Dement further contends that these microsleeps occur more and more often as we continue to deprive "total" sleep. Our efforts simply break up sleep into microsleeps. Subjects do not sleep for a single stretch of eight hours, but they sleep in bits and pieces throughout the day or night. Thus, the concept of "total" SD is not valid, and research on

total SD will not further elucidate the functions of sleep. By shattering methodological naiveté in SD research, Dement and Mitler have contributed to its progress. However, they fail to see that even such a blunt and imperfect tool as total SD can still be employed to reveal some secrets of sleep, if we are to couch and reconsider the findings of microsleep in SD research in the context of general sleep research. Instead of abandoning SD, we could scrutinize the characteristics of the microsleeps. Are the microsleeps mostly sleep stage 1, or are any other sleep stages involved? Under prolonged total SD of two or more consecutive nights, do microsleeps develop quickly into sleep stages 2 and 3, before the experimenter can intervene (8, 62)? Since there has been no study which recorded continuously the sleep-starved subjects with respect to electroencephalograms, electrooculograms, electromyograms and other variables, we have no idea what is the distribution of microsleeps over a 24-hour period. Similarly, we have no facts at all about the kind and duration of sleep stages, or the "recuperative power" of these microsleeps with regard to the removal of sleepiness and fatigue.

It is particularly instructive, however, to recall the fact that the SD studies conducted at Walter Reed Army Institute of Research and later at UCLA (65) suggest utter inability of even hundreds of microsleeps to ameliorate fatigue and sleepiness. The recuperative power of the aggregate microsleeps is not sufficient to sustain normal level of task performance. The limited amount of polygraphic observations in the UCLA study have shown that the subjects were almost constantly in a state closely resembling stage 1 sleep, that is, the state of *dormiveglia*. Yet these subjects experienced continual fatigue and relentless sleepiness. Polygraphically speaking, these subjects were experiencing sleep stage 1 saturation. Therefore, judging from the reported fatigue and sleepiness, shouldn't we conclude that sleep stage 1 can not be a substitute for sleep? If we were to control the duration of microsleeps, make them last a bit longer, would we be able to find microsleeps which are truly recuperative in the sense that they remove fatigue and sleepiness, shouldn't we conclude that sleep cuperative microsleeps include invariably certain s'eep stages, such as stages 3, 4 and REM, or would we find that they are always longer than certain minimal duration? These questions are related to those which we attempt to answer by studies of sleep stage deprivation and of short sleep stage deprivation and of short sleep or nap. Thus, a study of the microsleeps might resolve some fundamental questions about sleep.

Another feature of total SD may play an important role in sleep research. Total SD intensifies sleepiness, and extends the period of sleepiness which is usually short for normal sleepers (see Fig. 1). Fig. 1 shows a v'ew of spectral analyzed brain waves of a normal subject without SD, who goes through period of presleep or hypnogogic state, starting from 15 minutes after the bed-t'me and continuing until about 21 minutes. Because of the shortness of the span of time, it is very difficu't to explore the psychophysiological, biochemical and physiological nature of this sleepy state. With experimentally created intense and persistent sleepiness, total SD gives the researchers of sleep an easy multiple access to sleep onset. What is sleepiness? Is the sleepiness of normal subjects different from that of insomniacs, hypersomniacs, narcoleptics and patients suffering from other sleep disorders? If we suppress sleepiness by administration of amphetamine, would the sleep-starved subjects, now being no longer sleepy, be different from normal subjects (53)?

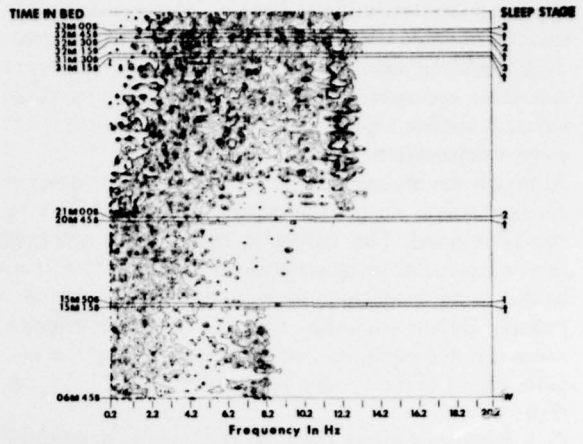

Figure 1:

A flat contour map of the C3-mastoid brain-wave spectral intensities of a normal subject without SD. The map shows the brain-wave frequencies on the X-axis, the elapsed time since the time to go-to-sleep along the Y-axis, and the spectral intensities as the equi-contour lines. This map can be read as a weather map or geographic map, showing the high's and low's. The subject was told to go to sleep at 06 min 45 sec. By 15 min 50 sec, he lost almost all of his alpha activity. The first sleep spindles appeared approximately at 21 min. This brain-wave record, together with the electro-oculograms (not shown), was scored independently from the spectral analysis, and the results are shown to the right of the map as W(ake), 1 (sleep stage 1) 2 (sleep stage 2) and 3 (sleep stage 3). The letter t represents a transition stage.

Dement and Mitler (14, 15) also maintain that REM sleep deprivation is not valid, because REM sleep is not a thing (such as the cerebellum or the kidneys) which can be removed completely. REM sleep is a confluence of many processes, such as bursts of rapid eye movements, low voltage electroencephalogram w'th occasional saw-tooth waves, electromyographic inh'bition, penile and clitoral tumescences, cardiovascular and respiratory irregularities, and others. Suppression of rapid eye movements does not necessarily prevent other REM sleep processes from continuing. An additional reason for the invalidation of REM sleep deprivation is that we can not deprive *all* of REM sleep by using the hand-awakening method, where the experimenter awakens the subjects at the earliest sign of rapid eye movements. Depending upon the alertness of

the experimenter, subjects have at least ten to twenty minutes of REM sleep over a night of REM deprivation. That could be enough to serve adequately whatever functions are usually fulfilled by much longer REM sleep. A similar argument can be offered to invalidate stage 4 deprivation.

Although the arguments of Dement and his colleagues are persuasive, sleep stage deprivation cannot be summarily rejected. The failure to confirm the supposed impairment after sleep stage deprivation (32, 45) may be due to the inability to measure the changes of appropriately chosen variables. Indeed, when we examine some selected variables, we can begin to see the tangible effects of REM sleep deprivation (24, 29, 54, 56, 60).

One feature of sleep stage deprivation is clearly predicted by Dement's methodological analysis; there are no dramatic effects. REM or stage 4 sleep is not vital for the individual's survival. Its absence is not a matter of life or death. Many studies on prolonged REM or slow wave sleep deprivation induced by drugs confirm this (1, 8, 50, 68).

Will our understanding of the functions of sleep be enriched by investigating reduced sleep or partial SD? Partial SD answers the question: how many hours of sleep do we need to maintain performance of daily routines?

There are two ways to approach this question: acutely reduced sleep or gradually reduced sleep. For acute sleep reduction, the habitual sleep duration is shortened over a single 24-hour period at the start of the experiment (25, 27, 63, 64, 67). This method of acute sleep reduction is accompanied by undesirable complications, as it imposes on the subject an immediate necessity to adapt to a longer sleep-wake cycle, and to altered daily routines. The gradual sleep reduction obviates such difficulties, since sleep duration is slowly curtailed step-by-step over a span of months, for example 30 minutes every two weeks from an initial 7.5 hours of sleep to 4 hours (30), so that subjects have ample time to adapt to new daily routines and a new life-style (20, 30, 31). A series of as yet uncompleted gradual sleep reduction studies has been reported by Friedmann and others (20) and Johnson and Naitoh (31). The subjects seem to tolerate a regimen of short sleep well, but only down to about 4.5 to 5 hours of sleep per day. Though functioning adequately beyond this point on 4 to 5 hours of sleep, they are unwilling to reduce their sleep further. Those who do display signs of sleep deficit, despite an ample time allotment for adaptation. In short, it appears that each subject has his own *sleep barrier*, below which he cannot shorten sleep without impairment. If the regimen allows one to sleep longer than this hypothetical sleep barrier, there is eventual adaptation to shortened sleep, whereas shorter amount below this sleep barrier means an accumulation of sleep debt.

This preliminary observation, together with previous studies on sleep reduction (25, 63, 64, 67), provides us with two insights: (1) sleep duration has shown a titration point in the sense that a switch occurs from eventual adaptation to an accumulation of sleep debt when sleep duration is less than the sleep barrier, and (2) as long as the minimal sleep duration is met, it does not matter how much more sleep is obtained in terms of task performance levels and subjective feelings. In other words, total sleep duration may not be as relevant as previously imagined in sustaining levels of task performances, as long as we exceed the minimal amount.

In previous studies of task performances, REM sleep was equally as effective as sleep stage 4 in restoring subjects from the degrading effects of total sleep loss (32, 39). REM sleep and sleep stage 4 are interchangeable to some extent and we may have to conclude that the sleep stages are not very important with regard to task performances. Now, total sleep duration is following suit.

After we have thoroughly satisfied ourselves as to the characteristics of the sleep barrier, perhaps we should investigate the question of how can we best arrange the sleep time (27), a question of the circadian effects on sleep. Taub and Berger (61) have already pointed out that a large proportion of the changes in human behavior, which have been customarily believed to reflect effects of SD, is in fact due to large circadian fluctuations. Similarly, Morgan (40) has observed an interaction between circadian influence and the effects of SD on task performances.

II. Non-Performance Measures of Sleep Deprivation

Because of the success of the Walter Reed Army Institute of Research studies (65) and the research of Wilkinson and his collegues (66, 67), SD research has inclined toward behavioral investigations, concerned with task performances, subjectve feelings, motivations, perceptions, memory and others, while neurological, biochemical and physiological modifications were sometimes shelved or treated only as processes to explain the observed behavioral manifestations.

This emphasis on task performances is unfortunate, because we might be misled to believe in an absence of SD effects due to the inherent robustness of human ability to perform under stress. An apt analogy can be drawn between task performance and body temperature. Body temperature is heavily controlled by many physiological mechanisms, such as sweating, shivering or increase or decrease of the peripheral circulation, so as not to change radically even under the extreme ambient temperatures and heavy exercise. An absence of radical deviation in body core temperature simply means that the temperature controlling variables are changing quite rapidly and the feedback is effective. Similarly, human task performance might not show any radical changes because many task performance-controlling factors are changing to assure behavioral stability (26).

In recent years, some SD studies have focused on the factors which control human task performances. Thus, it has been shown that total SD produces activation of the sympathetic adrenal medullary system and the pituitary adrenal cortical system (21, 22, 26, 36, 57). Earlier, Hasselman and others (28) reported a dramatic

increase in sympathetic adrenal medullary activity when sleep-starved subjects rode a bicycle ergonometer under low ambient temperature. The evidence, however, remains weak with respect to increases of urinary and plasma 17-hydroxycorticoids.

Another factor which helps to maintain adequate performances during SD is a metabolism step-up to meet the increased demand for energy. Research has suggested that total SD creates a perference to use lipids over carbohydrates as a source of energy (26), and also alters metabolic mechanisms (9, 10, 13, 62). The Czechoslovakian researchers, such as Brodan and Kuhn, found that the subjects experienced a significant decline in "physical fitness", as measured by the Harvard Step test, during the period of recovery from five days of total SD (9, 10). This may mean that the metabolic "debts" incurred, due to a stepped-up catabolism during SD, were "paid back" by an intense recuperative anabolism which used all of the resources during the recovery period. This limited severly the energies necessary for the Harvard Step test and recovery from such a strenuous test. Copes and Rosentswieg (13) have shown that the Harvard Step test for ninth graders suggests a decline of the "endurance" index during total SD. This decline can be interpreted as a reflection of an already-taxed metabolism in the children. The metabolic mechanism is no longer able to provide the surge of extra energy needed to achieve a quick cardiac recovery after the test.

Perhaps because of psychoendocrinal mobilization and altered metabolism during SD, there are changes in the hematological and immunological systems of the body. Two papers have reported changes in immunological reactions due to SD (11, 49). Also, an alteration in phagocytosis has been observed due to SD (48). Hematological changes due to SD have already been reviewed by Wilkinson (66) and Harris and O'Hanlon (26). An interesting hematological observation in rats has been reported by Drucker-Colin al. (16). They found that normocytic anemia will ensue in sleep-deprived rats under anticoagulant treatment, and for some, under additional treatment with para-chlorophenylalanine (PCPA). Under these conditions, rats lost many of their red blood cells and the hemoglobin was reduced to one half of the normal value. However, no hemorrhages were found to account for the loss of the red blood cells and hemoglobin. It is not known if aggravation of normocytic anemia by anticoagulant treatment could occur in men.

Another intriguing finding which is related to the study of Copes and Rosentswieg (13) has been described by Olree and others (47). By monitoring the level of heart rate increase to exercises, they trained healthy subjects, over a period of weeks, to be highly physically conditioned. Then, some of these physically conditioned men were asked to stop exercising, and they immediately started to lose their top physical condition at a slow but steady rate. Some others were asked to stay awake for just one night. Olree and others observed that the men lost their top physical condition very quickly after one night of total SD. It appears that a single night of total SD did not degrade the physical condition to below that level which the men had prior to the start of physical conditioning, but total SD had a telling effect on the men's ability to retain their peak physical condition.

III. Field Studies of Sleep Deprivation

In SD studies, laboratory demonstrations have usually dominated over field studies. Only recently there has been a renewed interest in evaluating whether men under field conditions will show performance impairment to a degree comparable to that experienced under laboratory conditions (31). Unfortunately, field studies have produced results which do not agree with those obtained by laboratory studies (31, 44). Why do SD effects fail to appear under the field conditions? There are perhaps three alternative ways to resolve the discrepancies between the field and laboratory studies.

Firstly, the failure to detect the SD effects under field conditions could be due to the use of insensitive and inadequate measures of the task performances. We would have to employ a sleep-loss sensitive measure, such as the supposed early decrement in reaction time tasks (37). Or, laboratory tasks which are known to be sensitive to SD could be modified for field use. There are small portable devices suitable for use in the field, which can administer a short pre-arranged task, score the task performances automatically, and then store the scores for later retrieval. In essence, devices would be analogous to a clinical thermometer, *i.e.* a *task thermometer*. The use of a clinical thermometer and a task thermometer would only minimally interfere with daily routines. The first step toward a task thermometer has already been taken by Wilkinson who has modified the five-choice serial reaction time task for field use. Secondly, the failure of field studies to detect SD effects may be the result of using group averages rather than extreme values. For example, the effects of short-term SD are small for the usual skills involved in driving a car. But, in certain hazardous situations, one lapse of short duration, or one severly affected individual can lead to a multiple traffic accident (38, 46). Some discrepancies between the field and laboratory studies may be resolved by greater attention paid to individual differences with respect to tolerance of SD. Thirdly, discrepancies may be a consequence of over-reliance on task performance measures. In field conditions, we should dispense with inherently stable measures of task performance. Instead, we should use some physiological (or other) non-performance measures. What would these measures be like? Most probably, they would be those variables which reflect a varying degree of sleepiness. Usually, sleepiness is detected by attenuation of brain-wave alpha rhythms, increased brain-wave theta activity, increased finger pulse volume, slowed heart rate, shallower slower respiration, constriction of the pupil (69), loss of the contingent negative variation (45, 65), inability to maintain coordinated muscle contractions (such as those needed to hold a piece of paper between the thumb and a finger, or to hold the head straight up

without bobbing, or to remain standing erect on the feet), and undulating slow eye movements. The most promising variables in the field conditions are the eye-movements and eye blinks (4, 58). Stern (58) and Angiboust (4) have reported that with sleepiness visual searches for targets decrease and eye-positioning to the targets becomes slower and inaccurate. Stern has observed also that eye blinks become slower with increasing sleepiness. These eye-movements can be elicited by a task of acquiring real-life visual targets, such as in the case of an army helicopter pilot (58), or by reading of written materials whose contents are directly relevant to the job at hand.

IV. Individual Differences in Sleep Deprivation

Although SD researchers have been interested in the individual differences in terms of susceptibility to SD (66), they have continued to collect SD data from a rather homogeneous group of subjects. In addition, few subjects are observed in SD studies. Putting these two facts together, one realizes that we are yet in no position to talk about research dealing with individual differences. A study of individual differences would necessarily require that we have a large sample of individuals, heterogeneous in terms of age, health, intelligence, personality and sleep characteristics.

Some recent studies have employed experimental subjects other than the young healthy college students and relatively young military volunteer subjects, *e. g.* human newborns (2, 3), 9th graders (an average age of 15. 2 years (13)), young alcoholics (an average age of 25. 8 years (62)), healthy middle-aged officers (an average age of 56 years (21, 22, 36)), and middle-aged alcoholics (an average age of 48 years (62)). Hopefully these studies mark the beginning of interest as to the modification of the impact of SD by age difference.

Although there are a few studies on the relation of personality to sensitivity to SD effects (44), the findings remain inconclusive. Similarly, we have only scanty evidence to support the view that certain individuals are born with physiological, biochemical and psychological characteristics which render them especially susceptible to SD effects. Can this supposed inborn sensitivity to SD be modulated by past experiences with SD effects, and by moment-to-moment physiological status?

V. Medically Oriented Research on Sleep Deprivation

I suggested at the very beginning of this paper that SD effects may pose some health hazards, and especially that physical ailments may ensue from repeated exposures to SD. On the other hand, SD has been found useful for combating depression (35, 51, 52).

Two significant contributions of the short-term SD to clinical medicine are concerned with aiding diagnoses in 1) some cases of sleep disorders, and 2) epilepsy.

SD helps diagnose the mechanisms which are responsible for certain sleep disorders, especially insomnia. To illustrate this point and also to simplify the further discussion, let us subscribe for a moment to the view of Jouvet, Mouret and others (33, 43), which assumes the presence of a properly balanced control of two opposing neurological organizations in regulating wake-sleep cycle, *i. e.* the waking and sleeping systems. Some patients experience no desire to sleep, that is, agripnia due to abnormally weakened sleep system. One patient with Morvan's disease, for instance, did not sleep for four months (19). Among those who are healthy in the wake-sleep cycle, we observe a growing dominance of the sleep system over the wake system as SD continues on for 24 hours or more, until the sleep system exerts its influences forcibly into the wake period, resulting in microsleeps. The fact that the subjects have a healthy wake-sleep cycle can be tested by looking at the recovery sleep records following SD (Fig. 2 and

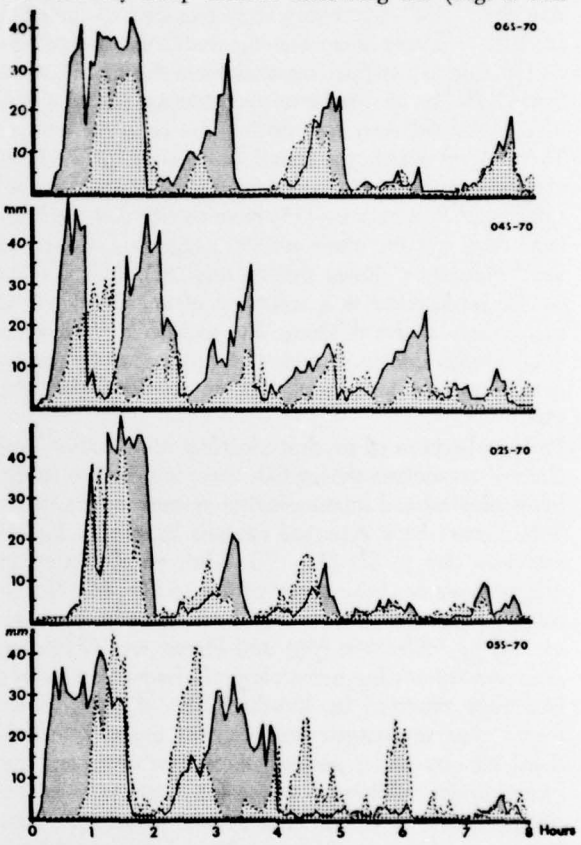

Figure 2:

Delta activities of sleep brain-waves of four subjects over eight hours of sleep. The X-axis shows the hours of sleep. The integrated delta activities are shown on the Y-axis in an arbitary unit proportional to uV^2. For details, see the text.

(41)). There are large increases in sleep stages 3, 4 or REM, and corresponding decreases in sleep stages 1 and 2. Fig. 2 shows an analog computer analysis of brain-wave delta activity (1—3 Hz) for four young healthy subjects before sleep loss, dotted histograms, and that after 60 hours of total SD plus two additional nights of stage 4 deprivation. During the recovery sleep, we see the increase in slow wave sleep due to both an early entry into slow wave sleep and longer duration of the slow wave sleep period, suggesting vigorous sleep system. A careful examination of the

patterns of recovery sleep from SD could reveal the strength of the sleep system. Thus, one could diagnose patients with insomnia by means of their recovery sleep, characterizing them as having a pathologically weak sleep system, an excessively strong wake system, and perhaps others.

SD is also helpful in the diagnosis of those suspected epileptics who exhibit normal brain-waves in routine examination (5, 7, 23, 55). These recent studies, together with those which have been reviewed by Naitoh (44), show that staying awake for one or two consecutive nights helps reveal abnormal discharges in the morning examination of electroencephalograms following total SD. Geller et al. (23) have shown that total SD can be performed even by small children at home, with encouragement from their parents, and further that total SD provocation remains effective for the children under anticonvulsant treatment. The fact that one night of total SD can reveal otherwise undetected focal abnormalities in the electroencephalograms makes SD a significant diagnostic aid (5). Unfortunately, we still do not understand the mechanisms of SD which provoke the abnormal discharges among suspected epileptic patients or among some suspected brain trauma cases. Blood glucose level seems to have no relevance to this increased proneness to seizure discharges. Also unsettled is the question of whether proneness towards abnormal brain-wave discharges is a direct consequence of the organismic state produced by SD itself, or is mainly due to increased sleepiness and fatigue in the morning following a sleepless night. It appears that SD will not produce abnormal discharges in brain-waves if the subjects are neurologically healthy. In other words, SD provocation has minimal false positives. On the other hand, SD provocation can result in some false negative cases. SD provocation could be useful for screening public transport workers, such as air-line pilots, bus-drivers and others.

Conclusions

With further methodological maturation, SD research is expected to contribute towards an understanding of the function of sleep. It can explore the nature of sleepiness, sleep onset and microsleep. A wider use of SD is anticipated also in clinical medicine. In addition, the SD research should continue to maintain a traditional inquiry into SD, where psychological, physiological and biochemical effects of SD are systematically catalogued to demonstrate potential health hazards imposed by SD. The results of such scientific inquiry on the SD effects could eventually lead to an improvement of the well-being of our society by warning of the dangers of excessive sleeplessness, and by showing how to keep a proper balance between wakefulness and sleep.

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Some recent studies on sleep loss reveal significant improvements in biochemical, physiological and psychological methods of assessment. Critical examination of these studies indicates altered research emphases, some newly developed research concepts, and a new awareness of the methodological complexity involved in the seemingly simple research tool, sleep deprivation. The present paper suggests that sleep deprivation research will remain important in advancing our understanding of the impact of sleep loss, and of the mechanisms for the need of sleep.

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